A CASE OF MULTIPLE NEURITIS, SIMULATING LANDRY'S PARALYSIS IN THE RAPIDITY, ORDER AND EXTENT OF PARALYSIS.[1]

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THROUGH the invitation of Dr. J. Madison Taylor, and by the kindness of Dr. S. Weir Mitchell, I report the following case. The simulation of the multiple neuritis in this man to the so called paralysis ascendens acuta (a disease rare and whose course and clinical features are not described alike by many authors) was a somewhat confusing element in the differential diagnosis, (if, indeed, any exist) at the time of admission of the patient to the Infirmary for Nervous Diseases on April 3d, 1893.

A. B., male, æt. 59 years. In the family history it is noted the relations were as a rule healthy and long lived. Two brothers had died in infancy, one as the result of an accident. A third brother succumbed to some bowel trouble at 27 years of age. Five sisters are all healthy at middle life. Father had died of old age; mother of apoplexy, æt. 68. No other neuroses or psychoses and no alcoholism.

The patient was always a healthy boy, had the ordinary diseases of childhood in mild forms. Boasted of his strength as a young man. Has never married and denies alcoholism or venereal disease. Thirteen years ago had inflammation of bowels.

Was wounded during service in the Civil War near outer malleolus of right leg and foot by a "spent ball," which was removed at the time, but the ankle bones were so crushed as to lay him up practically for two years. There was apparently no neuritis following the injury, but he has been lame ever since. Has never been able to do much manual labor since, and has not done any work for the past eight years. This excludes toxication (from the occupations' usual sources of poisoning) at least in the ætiology of the present trouble, which dates back seven weeks previous to admission to the hospital. The patient had been in good health and walking about up to February 16th, 1893. The next day he complained some of nausea, of "belching of wind," but no vomiting and no diarrhœa. Had a sense of malaise; later in the day felt weak in his legs and on waking February 18th, it was noticed he had some difficulty in articulation. His tongue felt thick. The legs, hands and arms had now become feeble. Patient walked about the house some, but later in the day the nausea recurring, a physician was summoned who pronounced the trouble "acute indigestion." The sister says he now had some fever. No flux or abdominal tenderness existed then, nor since. On arising Sunday, February 19th, (about 48 hours after the first attack of nausea) patient felt very weak. Said his knees felt as though they would give way, and when he got down stairs fell from inability to bear up the body weight by his enfeebled extremities. On attempting to walk again he fell a second time, after which he was assisted to the breakfast table where he ate a hearty meal, but by noon he became extremely weak all over his body, so much so that he could not feed himself and was carried to his room that night having considerably fever. There was no acute pain at any time and it did not cause pain to be lifted.

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[1] Read by request to the Philadelphia Neurological Society, December 26th, 1893.

There seems to have been no tenderness along the nerve trunks even from the first. He now remained in bed, the helplessness increasing rapidly in the inability to move legs, then arms and trunk, (although the power of aaticulation soon returned) until at the end of about 36 hours from the onset of this fulminating course of the paralysis he became almost completely diplegic. The fever gradually subsided, and his bowels moved normally and regularly as they had not done previous to present trouble.

He remained in this helpless condition and three weeks later fell from a chair he had been lifted into, (this was four weeks before admission to hospital) bruising the dorsi of both feet, it was thought. At any rate symmetrical greenish ecchymoses are found here at the first examination. There is also slight œdema of feet. He has no trouble with the kidneys, bladder or bowels, before or since the paralysis.

There has been no loss of sensation and there are no evidences of trophic change any place on the body.

Patient has had erections normally up to within a week of admission. The voice has gradually returned to normal, the only difficulty now being slight dypnœa and a tickling sensation in the throat causing hacking and expectoration of small amounts of mucus.

On admission too, the lungs are clear, (respiratory sounds faint) the temperature is 101°. Pulse, rapid and weak. No cardiac murmur. Is of fairly good physique. Liver and spleen of normal size and not tender in palpitation. The man's teeth are bad. No blue line. The bowels continue regular. There is slight tympanitis from abdominal paresis. Tongue clean. Appetite fairly good. Hearing good. Very soon the slight nervous cough lessened, and while he had no subjective pain, yet he bore an anxious expression, as one suffering from some very acute disease. He is entirely helpless. There is no face or tongue palsy, however, and the latter is protruded straight. Takes semi-liquids readily as fed by the nurse and there is no regurgitation. Can shrug his shoulders slightly. Can move toes and fingers a very little. Can flex knees slightly when supported on the bed. Makes slight quivering movement of abductors of thighs, not so of adductors. The muscles of the thighs, legs and arms, are "mushy" to the touch. The right thigh and leg are somewhat less in size than the left, due to old injury to foot and leg. The great toe of right foot is in position of hallux valgus, also probably induced by the gun shot wound of foot.

The sensation in both lower and upper extremities is unaltered as to touch, temperature or pain, excepting of a slightly tender point on deep pressure in the left popliteal space; none along other nerve trunks. There is some muscular soreness in the palms of hands and soles of feet, rather than a true hyperasthesia. The joints of fingers on both hands are sore on pressure, not to light touch. Not so in the toes. Tells pin prick any place readily. Tells point from head and there is no delay. Knee jerk is absent and not reinforcible by making a grimace, the only voluntary movement of any movement remaining. Plantar, cremasteric and abdominal reflexes nil. Dynamometer, r. o., l. o.. Jaw jerk is good, being the only reflex not affected, excepting the others of the head. The muscles react very slightly to blow of percussion hammer, especially so in flexors of forearms. After blow, muscles maintain a tetanic contraction, remaining "hard" a few seconds to half a minute. This is seen even in as small muscles of the hands as the first dorsal interossei, and more on right than on left side. The pectorals hump in like manner. Sphincters are normal. He sweats a great deal and the hands have "sour bread" smell, yet have been washed frequently.

The man's breathing is curious. There is little, if any movement of abdominal muscles or viscera. The respirations are feeble, hurried and seem to be costo-diaphramatic in type, the latter muscle not working satisfactorily, but producing jerky breathing.

Electrical examination by Dr. I. P. Willitts, was reported as follows, April 7th, four days after admission:

Faradic.—Two and a half inch coil, secondary; produces contraction in extensors both legs, most marked in tibialis anticus; same in arm extensors, flexors same.

Galvanic.—Five m. a. gives An Cl C = K Cl C in extensors of left leg; Seven m. a. gives K Cl C > An Cl C in extensors right leg, This shows marked quantitative change in all muscles affected with possible De R. commencing in left leg. This in seven and a half weeks.

Dr. De Schwainitz examined the eyes and reports, "with exception of sleight œdema of nasal edges of optic discs, there are no changes in the fundi. Vessels and form fields normal. Pupils respond to light and accommodation, but the left is a little the larger."

Thus, four days after admission and after this study of the case, (the crisis apparently over) Dr. Mitchell ordered continuance of diet confined to milk and vegetables largely, quinine sulph. gr. xii daily. Bowels to be kept open, and in active therapeusis alternating hot and cold baths to arms and legs b. d. There was absolutely no disturbance of his digestion as we found up to a week after admission and his diet was increased accordingly. Urinalysis negative. At this time also the temperature ranged from 100° to 101°. The paralysis which had seemed about to affect the medulla so seriously had not progressed. Examination of the spleen still revealed no enlargement or tenderness. The greenish spots of ecchymosis persisted over most of the dorsi of feet. No pain on pressure along nerve trunks. The only subjective symptom is a sense of formication in the extremities much relieved by the hot and cold baths. Muscular reactions to-day, (April 10th):—chest = normal reactions and humping on tapping with percussion hammer. Left arm: deltoid humping marked. Excessive humping in biceps both sides, the primary humping being feeblest. There is scarcely any reaction remaining in flexors of forearms. Both primary and secondary reactions very good in extensors of forearms, the humping continuing for a long time after the blow is struck. Also, he can shrug shoulders a little better. Can contract adductors of both thighs some but not enough to move limbs, The little gain in flexion and extension of fingers is less marked in left hand. Abductor muscles have gained some in power, but he can not move limbs out as he lies utterly helpless upon his back.

April 11th.—His temperature is 101°. Sleeps well, appetite good. The cough has ceased and lungs are clear. The plantar, knee, elbow, abdominal and cremasteric reflexes are still absent. There is faint fibrillary contraction on tapping anterior tibial groups and gastrocnemii both sides.

April 12th.—Patient had a good night's sleep. Today the slight movement of fingers is better in right hand, the first dorsal interossei muscle being the most active muscle on either side. Tapping this muscle produces marked fibrillary contraction. He has also recovered the use of abductors of thighs sufficiently to move limbs out a little as he lies on back. Takes nourishment well as fed to him.

April 13th. — Only change noted was that the fibrillary contraction did not exist while longitudinal contraction could be elicited a little in the anterior tibials. Longitudinal contraction was pretty well marked on tapping the gastrocnemii; a persistent fibrillary quivering continuing for some time after. The reaction to stimuli of both recti is fairly good. There is a slight plantar response easily exhausted on left, none on right side. The next day the muscles of arms were in better tone, excepting those of forearms. The right plantar reflex has returned feebly but the left could not be elicited. This vacillation of plantar reflex activity has been very marked. K. J. is absent. No tendo-Achillis reflex. The patient weighs 130 lbs., not much below his usual weight.

April 15th.—Dr. Kyle made cultures from blood taken from extravasations on dorsi of both feet, but with staining and high power no micro-organisms could be found. Thus the cause of the paralysis, if due to bacteria, was beyond our reach. That some infectious process was going on (possibly aptomanic poisoning) seemed inevitable, a blood count showing, out of 4,000,000 r. b. c. to c. m., only about 3,000,000 healthy cells. The remainder of the red corpuscles were irregular in form, crenated and many contained granular matter. Hæmoglobin estimate on admission being 68%, now was reduced to 50%. The white corpuscles were slightly increased. Surely all these were evidences of a profound dyscrasiac!

By the 18th of April, fibrillary twitching was everywhere lessened, and on the 20th, even more so, when too, the left plantar reflex had again reappeared. Measurements show right leg to be the smaller one, due to old injury stated, and not to unilateral acute wasting. But little difference existed in the thigh.

Massage and faradism was begun on the 23d of April, (10 weeks after beginning trouble) producing soothing and agreeable effect. He had no subjective pain. On the 19th an electrical examination had proved well marked reaction of degeneration existing in both legs, worse on right.

Galvanism: Left leg extensors, 6 m. a. An Cl C > K Cl C.

Galvanism: Right leg extensors, 8 m. a. An Cl C > K Cl C.

April 27th.—Sensation to heat, cold and touch perfect. Freezing part by ice and rhigolene had no effect on reflexes. The K. J. and elbow jerk remaining absent, the jaw jerk was well marked. On the first of May it was noted:— the right leg moves slightly more, and the right plantar reflex is increased in force. He breathes more by diaphragm, respirations less jerky, and liver now rises and falls with respiratory movements.

The same measurements of lower extremities obtained on May 8th as had been recorded April 20th, 1893.

May 20th.—All fibrillary contraction has disappeared. Cremasteric reflex has returned slightly.

Thinking of possible poison from arsenic in wall paper or from other source, a specimen of the man's urine was sent to Dr. John Marshall, at the University, who reported negatively. By the first of June the extension of paralysis was stayed, and he has made very slow improvement since as regards motion. He can now flex and extend right leg, adduct well, abduct slightly. The left leg can be adducted to lift foot 12 inches from bed, the patient being held on left side. Sensation seems perfect. If placed on right side can turn on to back.

Can shrug shoulders well. Can pronate, adduct and but slightly extend arm at elbow on both sides. Perfect control over bladder and bowels. Temp. 100°.

June 17th. — The note was made: No subjective pain anywhere. None on pressure over nerve trunks. No bad sores. Can adduct, abduct, supinate and pronate arm by shoulder muscles. Holds pencil in fingers for first time. Elbow jerks absent. Arm muscles respond faintly, left side best. Plantar reflex improved. The muscle jerks in legs are better on the right than on left side.

July 31st.—Faradic current gives no response in extensors both forearms. Three inch coil contracts extensors both legs.

Galvanic current: An Cl C > K Cl C in both forearm extensors = De R. present. This shows gain below and loss in upper extremities.

August 18th. —Slow gain. Diaphragm used more. Sensation to touch, temperature and pain normal. All motions improved. Joints of fingers are stiff from disuse. No K. J. or elbow jerk. Attempt at clonus in right foot. Leg muscle jerks good. Cremasteric and plantar reflexes normal. Temperature at night 100°, pulse 93° to 95°. By September 10th there was no decided change. The movements were slightly improved. Was now put on strychnia gr 1.40 t. d.

October 25th. — Sensation good. Motion much improved in hands and arms. Can touch nose with hand for the first time. No elbow or muscle jerks in arms. No K. J. or clonus. Plantar and cremasteric reflexes present. Muscle jerks in legs present. Can roll over on side in bed. Chest expands better in breathing. Controls bladder and bowels perfectly. Has erections normally.

November 1st.—Electrical examination by Dr. Willets:

Faradism.—Lost in all extensors forearms and legs, and in triceps.

Galvanism.—12 m. a. is necessary and gives An Cl C > K Cl C. in all the affected muscles. De R. present.

November 14th.—Patient's expression duller. Temp. 101°. Put on Ext. Thyroid gr. x. t. d., on empty stomach.

November 20th. —No change except that the temperature which has been running up to 99° and 100° has not been above 99°, and pulse is slightly more rapid. Reflexes same as last note. Can move all parts better.

November 23d.—Some dizziness when turning in bed. None when up on chair. Temperature not above 99° since taking thyroid. Pulse more rapid (increased from 110 to 120). Has same trophic change about great toe nail of left foot. No bad sores. Blood count is over 5,000,000 red blood cells, 75% hæmoglobin.

Status Præseus. — December 26th.—Patient was too helpless to be brought before you to-night. All muscles of upper and lower extremities are wasted and flabby. Interossei muscles, both hands, are very much wasted, making the hands passively claw-like. Can slightly flex, extend, adduct and abduct fingers, both hands, but the movements are clumsy. Complete wrist drop, both sides and on supinating forearms for him, he can not flex wrist, which can be done slightly, however, when not so impeded by gravity. Can put hands on head by action of shoulder muscles mainly, the forearms being flail-like. Muscles respond feebly to stimuli. Chest muscles are also flabby and pectorials act feebly. Jaw jerk, good response. K. J. and elbow jerk absent. Cremasteric and abdominal reflexes faint.

Can raise both legs high off bed; but the effort soon tires him, and the members drop flail-like on the mattress. Can adduct and abduct fairly well. Can flex and extend ankle and toes slightly. There is the characteristic ankle drop.

No tenderness along nerve trunks. No bad sores. The nails of first and third toes of left foot are now undergoing trophic change. All functions and emunctories fairly normal. Bowels moved daily. Urine also voided normally. Heart's action is weak. Chest expansion is poor.

To sum up—There first appeared nausea, difficulty of speech and of respirations. Then there was general weakness; finally sweeping like a cyclone the whole voluntary motor system below the head, beginning in lower extremities, extending rapidly to trunk and arms. These were preceded by malaise, fever, formications, but accompanied by no pain or tenderness along nerve trunks. All this took place too, within two or three days at least, placing a man in full vigor apparently, upon his back in utter helplessness, and from the history what would appear to have been grave bullar disturbance, the latter soon disappearing, although the vagi were weakened in their control over diaphragm, and the external respiratory nerves lost power over the serrati for weeks after the crisis was over. The adductors of thighs were more and sooner affected than the abductors, especially noted at end of first week after admission. The extensors or anterior tibial group were affected before (and more profoundly ever since) than were the posterior muscles of the legs. The extensors of forearms wasted before the flexors.

All these, with the rapid loss of reflexes, no early trophic changes, no history of alcoholism or other exciting cause, were a group of symptoms much resembling those of acute ascending spinal paralysis, so called. The age of the patient, the electrical inaction of the muscles; more in legs at first, then in arms, and, in July, '93, the gain (since April) in electrical response, of lower extremity muscles (even from the apparent degeneration of anterior tibial muscle of left leg) over those of the upper members, where degeneration had then taken place; and finally, the degeneration found in November, '93, of all affected nerve supply, (confirming the order of paralysis given above) are of course, *prima facie* evidence of the neuritis from the first and now existing.

Prognosis is unfavorable.—The increase in control and range of motion in degenerated states of nervus supplying the parts is always interesting, probably showing specialization of the remaining little or unaffecting fibres.

Strumpell says,[2] "there is usually no rise of temperature in ascending spinal paralysis." He thinks the symptoms must be due chiefly to functional disturbance from some toxic influence, and further states that "the condition of the reflexes and the rapid loss of electrical excitability in connection with the pains at the beginning seem to justify the hypothesis that the disturbance sometimes has its chief seat in the peripheral motor nervus, the disease then exhibiting the most acute form of infectious multiple neuritis," and finishes the pathogenesis as follows: "In other cases, however, the motor portions of the spinal cord, the lateral columns and the anterior gray cornua are, perhaps, chiefly affected." If there is recovery it is usually in a few weeks. Wood says,[3] "weakness, irregular formications, spots of numbness, weariness and discomfort," etc., are the prodromata of Landry's Paralysis. That paralysis begins in foot muscles, then legs, thighs, arms and in muscles of respiration. That the temperature is rarely above normal, and there is no pain (only discomfort, formications or slight numbness). No trophic changes. Sometimes marked anæsthesia. Usually none. "Œdema of skin" and marked sweating (as in this man) exist in some cases.

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[2] A Text Book of Medicine, p. 637.

[3] Nervous Diseases and Their Diagnosis, p, 51.

In Ross's thorough work[4] published this year in connection, Bury, most of the reported cases of Landry's paralysis are tabulated. He records 93 cases, and throwing three out for another category, states, that of the 90 cases remaining, 62 were in males, 23 in females; in the remainder, sex not being mentioned. That the majority of cases have occurred between the ages of 20 and 40 years, " but that persons have been attacked as early as three and as late as 67 years of age." He makes three stages, (*a*) invasion, (*b*) paralysis, (*c*) convalescence, and says there is, as a rule, no fever. Fifty-three cases in Ross's table are due to causes such as operate in ætiology of peripheral neuritis, viz.: diptheria, septicalmia, alcoholism, syphilis, malaria, infectious fevers, metallic poisons, etc. In 37 the causes were uncertain.

In 47 cases out of the 90, no mention is made of paresis of muscles supplied by bullar nerves.

In 15 out of this 47, it is merely mentioned generally that the paralysis pursued an ascending course, or that the extremities were feeble. In 20, it is stated the lower extremities were first or most profoundly affected than the upper extremities. In ten cases, the lower and upper extremities appear to have been attacked simultaneously. In one case, the lower extremities were alone affected, and in another, the upper extremities were attacked before the lower; 52 died, all in stage of paralysis, 38 made partial or complete recovery.

Morbid anatomy.—38 of the 52 cases were examined post-mortem, as were three cases dying of intercurrent attacks of acute disease in stage of convalescence, and one that died of abdominal cancer; making in all 42 autopsies. The case of Bauchet and the two cases of Schultze are the three out of the 93 tabulated cases Ross does not include as ascending paralysis *per. se.* in deference to Schultze where cases lasted six months and seven months respectively before death took place, and who prefers the name in such cases of "ascending atrophic paralysis." As no autopsy was made in the cases the true pathology was not found.

Ross says, " To sum up, the medulla and spinal cord were subjected to microscopical examination in 26 out of 42 cases in which an autopsy was obtained; in 14 of these cases both were found completely normal; in four more the lesions were situated in the medulla oblongata and clevical region—a localization which affords no explanation of the symptoms—and in the eight cases remaining, the changes described were so slight in degree that they can not be taken as evidence of myelitis. Some of the nerve-roots or peripheral nerves were examined in nine only of the 42 cases, and in three of them were found normal; while in the remaining six cases degenerated nerve fibres and other evidences of neuritis were observed. The balance, therefore, of the evidence afforded by morbid anatomy is largely in favor of the view that the essential lesion is to be found in disease of the peripheral nerves and nerve roots, rather than in the nerve centres."

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[4] On Peripheral Neuritis, A Treatise, p. 91.

In this case then—No metals, organic or other poison being found leaves us in ignorance of the subtle agent that must be the actiological factor.

The alterations found in the blood too, were the result of the same cause. It must have been aptomanic or toxine, and when the rapid advances in bacteriology reach the stage of isolating these toxic substances, we may hope too, for cure by the administration of antitoxines.

There being but five cases of Landry's Paralysis recorded in patients over 60 years, the explanation may be from the fact that tissue resistance is greater in the system that has become more or less immune to infectious processes through its having combatted many; whereas in the young the severe symptoms are less resisted for the contrary reason and the patient succumbs.

May not the pathological process in the quickly fatal cases be the incipiency of organic change which the accurate microscopic "techinque" is yet too crude to detect?

And may not this case of neuritis, running much the same course as Landry's Paralysis at first, be one tided over; the beginning organic change then having an opportunity to already continue.

Since the above report was made an abstract of a paper (by Kirilzew and Mamurowsky read at the "Jubilee" Nervenheil und Psyschologie in November, 1890.) was seen in the *Journal of Mental and Nervous Diseases*, 1891, xviii: 154. The paper was on "A Case of Landry's Paralysis with Autopsy." Patient was a locksmith, twelve days ill, died with well marked symptoms of Landry's Paralysis. Acute parenchymatous neuritis was found in nervus of feet, legs, forearms; to a slight extent the roots of clevical and lumbar enlargements of spinal cord. Cord—hyperæmia of gray cloudiness and atrophy anterior ganglion cells and accummulation of cells in and about central canal. Most marked evidences of inflammation were in the peripheral nervus.